From these facts they conclude that in the centrifugal transmission of excitations from the motor centres of the cortex of the cerebral hemisphere of a dog, there exists not only transverse interspinal paths of conduction, but also transverse intercerebral paths of conduction. GRACE PECKHAM.

PATHOLOGY (INCLUDING PATHOLOGICAL ANATOMY) OF NERVOUS SYSTEM.

Senile Changes in the Brain. KOSTJURIN. *Wiener Med., Jahrb.* 1886, Heft 2.

Kostjurin has examined a number of brains of old persons in order to determine the histological changes which present themselves in the various tissues in simple senile atrophy. He finds that the greater number of nerve cells in the cortex undergo a more or less marked degeneration with the production of pigment, and fat, and occasionally with the development of vacuoles. In the pericellular space numerous round cells are found in addition to the mass of detritus when the cell is entirely degenerated. The nerve fibres in the cortex are atrophied or reduced in number. The vessels of the cortex undergo an atheromatous degeneration, with the constant production of a connective tissue thickening in their walls. This may become so great as to obstruct the lumen of the vessels. The pigment deposit in the adventitia of the walls is also increased. In the place of the nerve-cells and fibres which have disappeared by atrophy there is found a slight increase in the neuroglia. On the surface of the cortex a large number of corpora-amylacea are found which may form a continuous layer covering the convolutions.

The intensity of these dangers depends less upon the age of the patient than upon the relative degree of loss of weight in the brain, so that they are more evident in light brains than in very old brains. They are sufficient to account for the mental symptoms of senility observed.

The Gulstonian Lectures on Spasm in Chronic Nerve-Disease. By S. J. SHARKEY. *British Medical Journal*, 1886, March and April.

By Spasm is meant excessive muscular contraction in defiance of the will or in excess of the intention. It may be persistent, the individual waves of contraction overtaking each other; intermittent, the individual waves succeeding each other with or without regularity; or co-ordinated, in which case groups of muscles are the seat of clonic spasms which are co-ordinated in such a manner as to produce some regularly recurring though involuntary movement. The motor mechanism through which all movement is produced consists of a cerebral part, viz.: the motor area of the cortex, and the pyramidal tract; and a spinal part, viz.: the an-

terior cornua of the spinal cord and the motor nerves. These parts originate and transmit all efferent impulses concerned in movement. Spasms may be produced by disturbances of the cerebral or of the spinal mechanisms, and by fine molecular changes which cannot at present be limited to any particular part of the motor tract.

I.—Spasm in connection with cerebral mechanisms.

Any injury to the cerebral motor tract between its cortical origin in the central convolutions and its termination in the anterior cornua of the spinal cord produces a degeneration in it below the seat of injury. This is followed by a tonic spasm in those muscles which are under the control of the injured fibres; increased tendon phenomena and rigidity being evidences of the increased muscular tone consequent upon the loss of cerebral control over the spinal centres. The partial or complete suppression of nerve impulses passing from the cerebral motor centres down the pyramidal tract may occur under a variety of circumstances, *e. g.*, acute fevers, anæmia, external debility, as well as organic disease. Its usual cause is a destruction of some part of the cerebral motor tract; *e. g.*, softening of the central convolutions in whole or in part, division of the internal capsule, sclerosis of the lateral column of the spinal cord. In these cases contractures are common. When the motor tract is pressed upon rather than destroyed tumors of a rhythmical kind appearing on voluntary movement are produced; and if the pressure produces finally a destruction of the tract, rigidity and paralysis supervene in the tremulous limbs. If the disease is within one hemisphere the symptoms are unilateral, but when it is situated between the basal ganglia and the medulla they are frequently bilateral. Bilateral tremor followed by paralysis and rigid contraction which finally become permanent, and succeeded in the last stage by occasional general tetanic spasms indicate in all probability a hard basilar tumor producing gradual pressure on the motor tracts where they lie in close proximity to one another. The diagnosis will be strengthened by the development of optic neuritis, and by paralysis of the cranial nerves. Since all these symptoms, including those of contracture and tetanic seizures, may be produced by pressure upon the motor tracts at any part of their course, it is no longer permissible to consider them as characteristic of cerebellar tumor as Hughlings Jackson supposed. They only occur in cerebellar disease when this is situated in the middle lobe and exerts pressure downward upon the pons and medulla—for where the lobe is destroyed by softening, no pressure on adjacent parts being produced, rigidity and spasms may be wholly wanting.

A number of interesting cases with records of post-mortem findings were cited to illustrate these points. Defective development of the brain involving the motor region and attended by congenital deficiency of the pyramidal tract, results in the condition of spastic hemiplegia, the mere absence of the controlling impulses, which in healthy subjects traverse the pyramidal tract,

being capable of giving rise to a spastic condition of the limbs. This condition, known as infantile spasmodic paralysis, may be unilateral or bilateral. Under these circumstances the spasm may be either mobile or fixed. If it is mobile the condition may be one of athetosis, rhythmical continuous movements of the fingers and hands or legs and toes, being kept up constantly; or the movements may resemble those of chorea, disseminated sclerosis, ataxia, or paralysis agitans. All the varieties of mobile spasm seem to result from a mixture, in varying proportions, of paralysis, spasm, and irritation, and their development depends upon lesions which interfere with the perfect functions of the motor centres and fibres, but which do not completely interrupt them. They may be situated in any part of the motor tract, and the variety of the movement does not depend upon the seat of the lesion, but rather upon the condition of the tract below the lesion. While the destruction of the basal ganglia has been attended by such spasms they are to be ascribed rather to an implication of the motor tract in the internal capsule than to the disease of the ganglia, since the latter may be entirely latent. A case was related of total destruction of the caudate and lenticular nuclei on the left side, with descending degeneration in the inner third of the crus and in the pons, but no changes whatever in the medulla or cord. In this case two attacks of hemiplegia had occured, from which the patient had recovered completely several years before his death, no paralysis or rigidity remaining. Our present knowledge of anatomy, physiology, and pathology does not justify us in concluding that there is any efferent motor connection between the brain and the spinal cord except the pyramidal tract, and this has no connection with the cerebellum or basal ganglia, and its fibres are nowhere interrupted by entering ganglionic centres, but pass straight to their several destinations in the anterior cornua. If the tract be interrupted at any point in its course in the spinal cord all the spinal centres which are under voluntary control below the point of interruption will be thrown into a state of hyperphysiological activity. The symptoms of disease in the lateral columns of the cord are the same whether it follow cerebral or spinal lesions. They differ somewhat according as the pressure made upon the lateral columns is from without inward or from within outward. When a tumor grows in the spinal canal outside the cord, it may produce but few symptoms until it presses the cord against the resisting walls of the canal; but after this has taken place the course of the disease is very rapid, as the cord is quickly flattened by the constantly increasing demands for growing space which are made by the tumor. When a tumor arises, on the other hand, within the spinal cord, it disturbs its functions even from the very commencement; but as the nerve substance is elastic and allows gradual stretching without serious interference with its functions, a tumor may go on growing for a long time before it produces striking pathological phenomena, either by pushing the cord

against the bony walls of the spinal canal, or by exhausting the elasticity of the membranes which envelope it. In a case of tumor pressing upon the upper dorsal spinal cord from without the symptoms were in the order of time, weakness of the legs in walking; increasing weakness of the legs, accompanied by cramp and shooting pains; paraplegia, cramp, and involuntary flexing of the legs; slight numbness and long-continued attacks of muscular spasm in the lower extremities, and finally persistent rigidity. In a case of tumor within the cord, in the cervical region, the symptoms were partial loss of power and sensation in the arms and hands, and some tremor in them when used; not till twelve months after the beginning of the symptoms were numbness of the legs, loss of power, and incontinence of urine, with exaggerated reflexes, observed; and these were referred rather to a hemorrhage in the dorsal region than to the tumor in the cervical region. The contrast between the two cases is striking. Primary lateral sclerosis characterized by weakness of the limbs, tremulousness, jerkings, and rigidity is easily recognized if it is not complicated by sclerosis in other tracts than the pyramidal columns. When the posterior columns are also involved the symptoms may be obscured by those of locomotor ataxia.

II.—Spasm in connection with spinal motor mechanisms.

By the spinal system it is intended to include all the nerve centres with their efferent and afferent nerves which occur between the central ganglia of the brain and the termination of the spinal cord. (1) Spasm produced by diseases of the efferent spinal nerves. In the majority of cases where muscular spasm causes distortion it is the healthy muscles which are actively concerned, while the diseased muscles are for the most part passive, *e. g.*, main en griffe, of progressive muscular atrophy; deformities of pachymeningitis cervicalis. The rigidity occuring in alcoholic paralysis is of this kind, the healthy muscles overcoming their weakened opponents, which are usually the extensors. The same is occasionally seen in lead palsy. In all such cases the existence of normal electric reactions in the rigid or contracted muscles reveals the true condition present. In some cases, however, it is the shortening of diseased muscles which produces distortion. Here the diseased muscles are rigid; when attempts are made to extend them passively they do not relax under anæsthesia, and they have usually lost their electric contractility. Chronic muscular spasm is quite the exception in peripheral nerve disease, although occasional spasm may occur from this cause. Direct irritation of motor nerve filaments rarely causes contractions in the muscle, though it is often severe enough to produce paralysis. In this connection Weir Mitchell's opinion was quoted, viz.: "The symptoms in disease of the peripheral nerves affect at first rather the sensory sphere than that of motility. We have pain and anæthesia; or hyperæsthesia, but not, as a rule, convulsions. In certain cases the nerve wound, in place of causing primary loss of motility, occasions either sudden muscular contraction, followed by instant

loss of power, or in very rare instances, long-continued spasm. Tonic contraction of muscles are occasionally met with at a later stage of these injuries, but are perhaps amongst the rarest of the secondary symptoms."

(2) Spasm produced in a reflex manner by disease of afferent nerves. Such spasm is to be referred to a hyperphysiological activity in the spinal centres rather than wholly to the stimuli applied to the afferent nerves. Muscular contraction of a reflex kind varies much in degree in different individuals and in different states of health, although the exciting stimulus may be the same. Hence it is probable that the injury which supplies the stimulus to the sensory nerve in these cases though apparently the principal agent, is really so in many instances only from a particular point of view. Had the same stimulus been applied to the same nerve when connected with a healthy and stable centre no spasm would have ensued. Reflex spasm no doubt occurs, but how frequently it does so, or how far the afferent or efferent nerves or nerve-centres take the leading part in its production are points which can scarcely be estimated. The condition is reflex when sudden pain in a part succeeds spasm. Persistent reflex spasm may be due to irritation from joint disease, as Charcot has shown, the joint being rigid until the patient is anæsthetized. The muscles normally contract slightly when they are stretched by voluntary contraction of their opponents. This reflex contraction may under certain conditions—especially overexertion—become excessive, giving rise to sudden movement of the limb in a direction just opposite to that intended. This is very probably the cause of the tremor in the direction of the movement seen in disseminated sclerosis. It often follows sprains, in which certain muscles are overstretched. Interesting cases illustrating this condition were related. What is really developed in these cases is an involuntary neuro-muscular habit, and it is probably associated with an abnormal state of the nerve-centres as well as with disease of the afferent nerves. Hence it often yields to the use of bromides.

III.—Functional spasms may be referred to molecular changes, not visible by the aid of microscope, in either the cerebral or the spinal parts of the motor tract, and therefore they can be classified in the same manner in which spasms from organic disease have been. (1) Functional disorder of the cerebellar-motor mechanism is seen in hysteria, hemispasm, either fixed or mobile, being not infrequently observed. There is this difference between the hysterical conditions and those which are seen in gross disease : the leg is affected most, the arm less, and the face not at all. Apart from this the spasms are similar. In functional diseases there is simply removal of the voluntary impulses which in health pass down the pyramidal tract. The muscles and nerves remain practically healthy, but rigidity and increased tendon reflexes are present, just as in those cases in which there is a congenital defect of the motor tract. Absence of voluntary impulses is enough to give rise

to the phenomena in question, and hence it is legitimate to conclude that suppression of the functions of this tract is the cause of functional hemispasm. Monospasm, athethosis, and allied mobile spasms which occur in hysteria are to be explained in the same way.

There is another class of functional spasms which, I believe, have their origin in suppression of the functions of certain portions of the pyramidal tract, viz.: professional hyperkineses, *e. g.*, writer's cramp, pianist's cramp, etc. When a voluntary effort is made to perform a certain act, spasm of the muscles involved occurs, and prevents further effort. Before this stage of the affection is reached, great fatigue often accompanies endeavors to work. What has occurred is probably that after long-repeated acts of the same kind, that part of the pyramidal tract which is used becomes fatigued, and its functions are partly suppressed, so that a condition of "latent contracture" of the muscles over the voluntary actions of which it presides is developed; the lower centres are "let go," as Jackson says, and are in a state of hyperphysiological activity.

(2) Functional disorder of the spinal motor mechanism is also observed. Of course deformities due to atrophy of some muscles, and contracture of their opponents, or to shortening of diseased muscles, cannot be called functional, nor should we expect to find that irritation of the afferent or efferent nerves would give rise to functional spasms, since it rarely causes spasm when it goes far enough to produce gross changes in the nerves. The nerve-centres of the cord may, however, develop a state of hypersensitiveness and of hyperkinesis without gross lesions, and hence functional spasms from this cause are not rare. There can be no question that one of the peculiarities of nerve-centres in hysteria is their abnormal irritability, so that slight afferent impressions give rise to muscular acts which pass with great ease into neuro-muscular habits. Such a condition is well exemplified in cases due to irritation. The patient sees a certain form of muscular spasm, and the idea produces the same in her. Interesting cases of this affection were related. Persons may have those peculiarities of their nervous system which are usually embraced under the term hysteria, without ever presenting striking emotional or other tendencies which are wont to call attention to the existence of the disorder. The first evidence of the latter may be the sudden supervention of spasms from a very slight external injury. In any case, the more the cerebral control retires to the background, the more likely are spasmodic contractions to come to the fore-ground. Hyperexcitability of the spinal-centres without a diminution of cerebral control occurs under the use of strychnine; hence it is not impossible that molecular changes may produce the same state, giving rise to spasms on any voluntary action.

In the comparison between the results of gross disease on the one hand, and molecular, or so-called functional alterations, on the other, the pyramidal tract stands prominently forth as the

great offender in the production of muscular spasm. Its action is indirect, it is true, as it only loosens the reins of the spinal centres, which it should keep well in hand. Still it rules the situation. Spasm rarely ensues directly from injury or disease of the peripheral motor-nerves, and, although it frequently results from reflex causes, it is very likely that the spinal-centres are, in a considerable proportion of cases, more at fault than the afferent nerves. Considering how late the pyramidal tract develops in man, and what a high pitch of evolution it represents, it is not to be wondered at if it is one of the first parts to suffer in the process of dissolution. It is important to remember that exhaustion in one area of the motor system seems to affect the whole system, disturbing that equilibrium which is necessary to normal action.

M. A. STARR.

On Hysterical Anuria with Secretion of the Urine by the Stomach, and Experimental Researches Made in Hysterical Anuria in Relation to Uræmia. By Dr. EUGENIO ROSSONI. *Rivista Clinica*, October and November, 1885.

The writer gives the report of two extreme cases of hysterical anuria in which the most careful observations were made. In one of these the patient vomited a fluid which in every way resembled the urine, which at times corresponding to the menstrual epoch, the menses being absent, was tinged with blood. Space would be wanting to go into the details of this extremely elaborate article, but we give the conclusions arrived at, including the results of experimentations with pilocarpine and urea. Anuria is a phenomenon which is undoubtedly more often manifested in hysteria than is generally believed. Hysterical anuria is found in a peculiar state of the physical organism, which may be recognized by a retardation and limitation of the general nutrition, together with a state of neurasthenia of the nerve-centres, more especially those which influence the secretory activity of the kidneys.

An hysterical person with anuria may live a long time without manifesting grave uræmic symptoms, this depending on the morbid condition of the general nutrition, and not of necessity giving rise to vomiting, to compensate for the suspended functions of the kidneys. This vomiting may be lacking entirely.

Not every kind of vomiting which may be manifested during hysterical anuria should be believed to depend on the anuria.

In hysterical persons, with suspension of the function of the kidneys, may be found in their stomachs a fluid more or less abundant, which has all, *ad literam*, the physical as well as the chemical properties of normal human urine.

The activity of this urine-like secretion does not stand in inverse relation to that of the kidneys.

The urine-like secretion of the stomach may be suspended for a time more or less short, without a contemporaneous return of the renal secretion, which may remain absent a considerable time longer.

The existence of hysterical anuria should not be denied when, in a given case, vomiting of urinous fluid is lacking.

Given a case of hysterical anuria in which the functions of the kidneys may be suspended, pilocarpine will bring about a temporary activity.

Pilocarpine may have an active effect in exciting renal secretion of human beings.

In some hysterics with anuria pilocarpine may determine the secretion of a saliva having all the physical and chemical characters of normal human urine.

Urea introduced artificially into the circulation in an hysterical patient with anuria and without secretion of urine from the stomach, acts perniciously—determining uræmic attacks.

Urea by itself may contribute to the development of uræmic symptoms; may be introduced artificially into the system, in quantities of sixteen grams in a short time, in hysterical subjects with anuria, if there is active urinary vomiting.

There does not exist any identity between hysterical anuria and the uræmia of nephritis and of animals with extirpated kidneys or ligated ureters. GRACE PECKHAM.

A Contribution to the Doctrines of the Innervation of Movements of Expression. By Dr. P. ROSENBACH, of St. Petersburg. *Neurol. Centralbl.*, 1886, No. 11.

It is a well-known fact that in facial paralysis of central origin, muscles which cannot be moved by voluntary effort, may contract in obedience to reflex impulses. By a comparative study of clinical and pathological data, Nothnagel concludes that this condition could exist only in case the optic thalamus and the fibres of the corona radiata joining the thalamus and the hemisphere had not been interfered with. Nothnagel's views have been corroborated by recent investigations of Bechterew. Rosenbach now reports a case in which the ordinary condition of things is reversed, for in this instance the facial paresis is not made evident until the patient begins to laugh.

The patient, a women æt. thirty six, had an attack of left hemiplegia (sudden onset without loss of consciousness) ten months ago. When the facial muscles are at rest, there is only the slightest trace of paresis of the lower facial muscles. In speaking, both halves of the face can be moved equally well. The left half of palate is lower than right half. When the patient attempts to laugh, *the left half remained altogether passive*, the left naso-labial fold disappeared, and the mouth was drawn to one side. The examination revealed also *left bilateral hemianopsia*, anæmia, and organic cardiac lesion. The occurrence of hemianopsia, together with the loss of movements of expression, leads the author to locate the lesion in the optic thalamus. No autopsy; and yet we think the auhor was justified in publishing the clinical data.

Tabes with Presence of Knee-Jerks. Discussion in the Berlin Society for Psychiatry and Nervous Diseases. After a report in *Mendel's Neurol. Centralbl.*, No. 10, 1886.

Westphal read a paper on cases of tabes dorsalis in which the knee-jerk was not at all times absent.

Bernhardt observed that he also had seen such cases, and referred furthermore to a case of disseminated cerebral tumors, in which the knee-jerks were absent. Mendel found the knee-jerk absent in the earlier stages of tabes, and either on one side or on both. He called attention to a variety of cases in which the knee-jerk is not obtained, and instanced a case of tumor of the cerebellum in which the knee-jerk was absent, without any changes having been found post-mortem in the spinal cord. The reason of this loss of patellar tendon reflex, he is not able to explain.

Thomsen referred to ten cases of cerebro-spinal and tubercular meningitis, in five of which the knee-jerk was absent; the spinal cord was examined in all these ten cases, but no histological changes were discovered. Thomsen thinks that in such cases the peripheral nerves should be examined. Westphal states that he never believed the knee-jerks to be absent in tabes only. This symptom occurs in cerebral diseases as well, in which there is considerable diminution of muscular tone. Jendrassiks' method of eliciting the knee-jerk can be explained by the fact that increased (involuntary) innervation of all the muscles, including the *quadrieps femoris*, increases the tone of the muscles, and can thus excite the knee-jerk, which could not be obtained while the leg is at rest.

Contributions to the Pathology of Tabes. OPPENHEIM and SIEMERLING. Read at the Berlin Soc. for Psy. and N. D., and reported in *Neurol. Centralbl.*, 1886, No. 11.

At the instigation of Prof. Westphal, the authors made a careful examination of the condition of the peripheral nerves in cases of tabes dorsalis. In order to have a reliable basis for comparison, they examined the peripheral nerves in a large number of individuals who had died of other than nervous diseases. The result of this study was a conviction that lesser degrees of degeneration occur in cases of infectious or toxic disease, and as the result of a general marasmus. Extreme nerve-degeneration, however, was observed only in cases of multiple neuritis, either of tubercular or alcoholic origin.

In the majority of cases of tabes dorsalis, the sensory nerves and their branches had undergone excessive changes, such as were found in no other cases but those which had presented the clinical symptoms of neuritis. This excessive degeneration was found (in seven cases of tabes) in the branches of the N. saphenus major, in the smalll branches of the peroneal which supply the skin of the toes, and in the digital branches of the ulnar nerve. Similar changes were noted in the X., and in the recurrent laryn-

geal nerve of persons who had been afflicted with gastric and laryngeal crises. In every instance the changes in the sensory nerve were found to be in excess of those in the larger branches of peripheral mixed nerves.

The authors call attention to a special form of degeneration in many cases of tabes. This was as follows: On cross-section the perineurium is seen to be considerably thickened; the nuclei very numerous. Between the perineurium and the nerve-fibres there is a vascular tissue. The blood-vessels in this layer of tissues are for the most part obliterated, and their walls have undergone sclerotic changes. The blood-vessels are so numerous that the nerve-substances appears to be surrounded by them. This condition represents an interstitial neuritis or perineuritis. This perineuritis was observed in persons in whom tabes was complicated by other diseases.

The posterior roots and the spinal ganglia were diseased in some cases of tabes. As for the connection between the disease of the posterior columns and the degeneration of the peripheral nerves, the authors deny any connection of the sort, either condition not being necessarily accompanied by the other. It is but one step further to infer that the sensory disturbances, including ataxia, and the phenomenon of delayed sensory conduction, are due to these changes in the peripheral nerves. The changes in the vagus and recurrent laryngeal will account for the gastric and laryngeal symptoms. It is worth adding that the nuclei and roots of the vagus, and the post. longit. fasciculus of the oblongata, were normal in cases in which the peripheral affection existed. With regard to the condition of the spinal cord, the authors only report that they have frequently observed atrophy of nerve-fibres in the columns of Clarke. The usual changes are not referred to. B. S.

The Effect of Extirpation of the Ovaries upon Nervous Symptoms. SCHMALFUSS. *Arch. fur Gynæcologie*, xxvi., 1.

In a valuable article upon this subject the author sums up the results obtained in Hegar's clinic at Freiburg. Only those cases of extirpation of the ovaries are considered in which nervous symptoms had predominated, and which had remained for some time under observation after the operation. Anatomical changes were discovered in the large majority of the ovaries cut out. Prior to the operation in all cases a long course of general treatment had proved unavailing. The thirty-two cases reported are divided into three groups. In the first group are placed cases in which the nervous symptoms had been confined to the sacral and lumbar plexuses and their distribution. In the second group cases are placed in which other nerve regions had been affected. In the third group the cases of general neurotic condition with symptoms of reflex disturbance and neurasthenia are classified. In all these cases, except two, a decided diminution in the num-

ber and degree of the nervous symptoms followed the operation. In twenty-four cases an entire cure resulted; all the nervous symptoms disappearing sooner or later after the extirpation. In six of the eight remaining cases a decided improvement was produced. An explanation for the failure in the two cases mentioned is found in local conditions which the operation necessarily failed to reach. M. A. S.

MENTAL PATHOLOGY.

Insanity in the United States. Dr. A. D. WRIGHT (Conference of Charities, Eleventh Session, October, 1884) gives the following table of the proportions of the insane to the sane in the United States:

NEW ENGLAND STATES.

Maine, one insane to every	421
New Hampshire, one insane to every	329
Vermont, " "	327
Massachusetts, " "	348
Rhode Island, " "	404
Connecticut, " "	361
Total population, 4,010,629; one insane to every	359

MIDDLE STATES.

New York, one insane to every	362
New Jersey, " "	470
Pennsylvania, " "	516
Total population, 10,496,878; one insane to every	424

INTERIOR STATES.

Ohio, one insane to every	439
Illinois, " "	600
Indiana, " "	558
Michigan, one insane to every	586
Wisconsin, " "	521
Total population, 13,091,477; one insane to every	610

NORTHWESTERN STATES.

Iowa, one insane to every	639
Minnesota, one insane to every	681
Kansas, " "	996
Nebraska, " "	1,095
Total population, 3,853,886; one insane to every	750

SOUTH MIDDLE STATES.

Delaware, one insane to every	740
Maryland, " "	504
Virginia, " "	627
North Carolina, one insane to every	690
Total population, 3,993,866; one insane to every	610

SOUTH INTERIOR.

West Virginia, one insane to every	630
Kentucky, " "	592
Tennessee, " "	642
Missouri, " "	655
Total population, 5,977,886; one insane in every	629